Abstract

Disclosed is a mutant human α-synuclein with decreased ability of forming aggregation. The mutant human α-synuclein of the invention is able to inhibit aggregation of the wild type human α-synuclein, Ala53Thr mutant human α-synuclein or Ala50Pro mutant human α-synuclein, thus is useful for investigation of pathology and treatment of Parkinson's disease and for research and development of gene therapy. Also disclosed is a partial structure peptide of human α-synuclein comprising amino acid substitutions as taught by the invention.